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DRY-ROT OF SWEDES AND TURNIPS.

SEED SHOWN TO BE PRIME CARRIER OF THE DISEASE.

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[*The following abridged article has been prepared for the Journal by Dr. Cunningham from a technical bulletin treating fully his investigation and conclusions on this disease, which has just been issued by the Department of Agriculture under the title "Dry-rot of Swedes and Turnips: Its Cause and Control."—Ed.*]

DRY-ROT is to-day the most serious disease attacking swedes in New Zealand, for in a season favourable to its spread it may destroy from 50 to 100 per cent. of the crop. On turnips its effects are generally much less severe, but even with this host the loss may amount up to 40 per cent. Infected bulbs become a total loss; consequently the direct loss from this disease is much greater than is first apparent, for loss of the greater part of the swede crop seriously affects the carrying-capacity of the farm.

Appearance and Effect on the Hosts.

The appearance of the disease varies considerably according to the part of the plant attacked; therefore this will be considered in relation to each part.

Leaves: Small circular spots, slightly sunken below the surface, tobacco-brown or ashy-grey in colour, appear on leaves, first becoming noticeable on the cotyledons, and present thereafter until maturation of the bulb. In these spots small black bodies, the fruiting structures of the causal organism (termed pycnidia), soon make their appearance. These bodies are frequently arranged in concentric circles.

Bulbs: Early infection of the bulb occurs in the region of the neck, and on account of frequently being hidden beneath the leaves the lesions are often overlooked. The lesions produced are at first small and leaf-green in colour; they soon become slightly sunken, and turn grey or brown. In small bulbs the disease soon spreads through the tissues, causing death of the bulb. At this stage the lesions are few, but often of considerable size. Later they become more numerous and are often scattered over the whole surface of the bulb, though rarely below ground-level. They do not penetrate so deeply into the flesh; consequently, though more numerous, they seldom kill the plant outright until later in the season, when the lesions tend to merge one with

another. Such a condition is followed by death of the bulb; it does not tend to rot, however, as do those infected earlier in the season, but dries out and assumes a "mummied" appearance. The appearance of the disease is frequently masked by the presence of another pathogen, which sets up a soft rot.

Pycnidia are often abundant in lesions on bulbs, but are by no means characteristic, as they are just as frequently absent.

Stems: On stems of cabbages and flowering-spikes of swedes and turnips are produced lesions brown or grey in colour. On cabbages infection is frequently followed by death of the plant, but on spikes small depressed areas only are produced, the spike rarely being killed unless the lesions are very numerous.

Siliquas: On the siliquas (seed-pods) characteristic brown or ashy-grey lesions are found. In the interior of the pod a flocculent mycelium is often present, extending to and surrounding seeds in the vicinity of the lesions. The significance of this seed-infection will be made clear later in this article.

Life-history of the Causal Organism.

Dry-rot is caused by the fungus *Phoma lingam*. In New Zealand it has hitherto commonly been attributed to *Phoma Napobrassicae*, but in his technical bulletin the writer has shown this to be a synonym of the former. *Phoma lingam* has been found in the Dominion on swede and turnip bulbs, leaves, spikes, siliquas, and seed; and on leaves and stems of cabbage, but not on kohl rabi, chou moellier, rape, or wild turnip, nor other weeds belonging to the family Brassicae.

Experiments conducted in this Laboratory have proved that the disease is perpetuated by means of resting mycelium in the seed-coat. When the seed germinates this structure (termed the testa) is frequently carried upwards on the tips of the cotyledons. Before the latter expand the fungus grows out from the testa into the cotyledon tips, there producing a small lesion (brown in colour, and therefore readily noticeable) on one or both cotyledons. This lesion is formed by the hyphæ of the fungus penetrating into and destroying the cells of the leaf. These turn brown and die, and among them knots of hyphæ are produced which eventually take the form of flask-shaped receptacles or pycnidia.

In the pycnidia very many one-celled colourless spores are produced. The pycnidium, as has been shown, is embedded in the tissues of the leaf, and opens to the exterior by means of a small apical pore (termed an ostiolum). The spores are embedded within the pycnidium in a mucilaginous matrix, which is strongly hygroscopic—that is, it has a strong attraction for water. Consequently in wet or humid weather the mucilage absorbs water to such an extent that it swells and is forced through the apical pore of the pycnidium as a small gelatinous tendril, carrying with it the embedded spores. In this manner spore-discharge occurs, but as such is possible only in the presence of abundant moisture it will be seen that discharge is possible only during wet or humid weather, and cannot occur during dry weather. The tendril is soluble in excess water; consequently in wet weather it becomes of a liquid nature, and is washed down the leaf or spattered in rain-drops

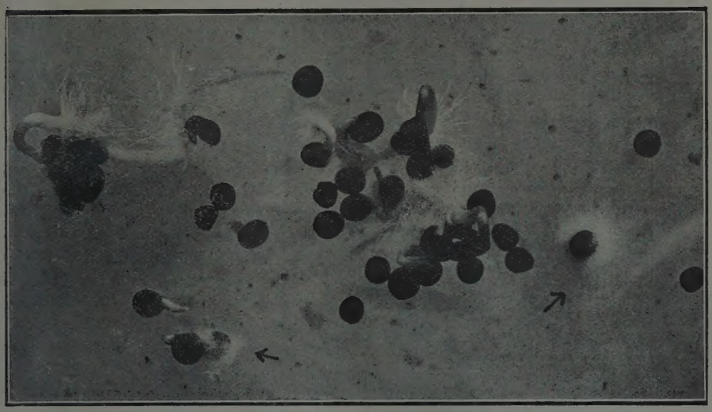

FIG. 1. DRY-ROT FUNGUS (INDICATED BY ARROWS) GROWING FROM SEEDS TO CULTURE MEDIA THREE DAYS AFTER PLATING. × 5.

FIG. 2. GERMINATING SWEDE-SEEDLINGS, SHOWING MANNER IN WHICH TESTAS (SEED-COATS) ARE CARRIED UPWARDS ON THE COTYLEDON TIPS. × 2.

Arrow points to dry-rot lesion on cotyledon of plant on the right.

[*Photos by H. Drake.*

to leaves of contiguous plants, each drop carrying very numerous spores. If a dry spell prevails, the spore tendril dries out and becomes firmly fixed to the surface of the leaf (or bulb, as the case may be); consequently, until moisture is again present, spore-dispersion is not possible. Spores may remain alive in dried mucilage for over six weeks, but if the mucilage is removed they live for only five to ten days or less.

It will thus be seen that the disease is carried to the field in the seed, and therefore that diseased seedlings will be present in the field a fortnight or less after sowing, their number varying according to the proportion of disease present in the seed. This proportion varies greatly, in some lines being about one infected seed in a thousand, in others as high as five seeds per hundred. If dry conditions are met with after sowing, or if the seed is sown late in the season (say, January), the probability is that these initial infection sources are considerably lessened, and possibly in extreme cases eliminated.

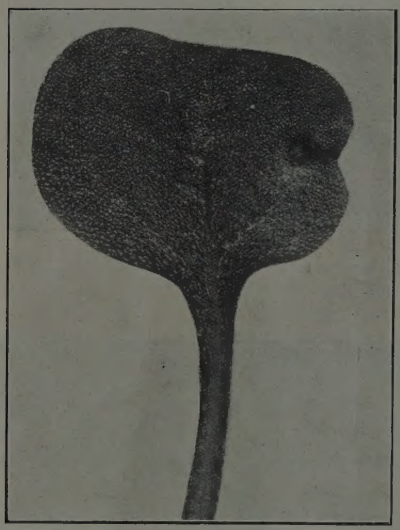

FIG. 3. SEEDLING LEAF OF SWEDE TWELVE DAYS AFTER GERMINATION, SHOWING LESION PRODUCED BY THE DRY-ROT ORGANISM. × 5.

[*Photo by H. Drake.*

The fungus spreads by means of the spores to other leaves of the same plant, and to other plants in the vicinity. Apart from forming lesions on the leaves it does little damage, however, for as yet no bulbs are formed. Further, it has been found that at this stage the disease seldom spreads beyond the initially infected plant, or beyond those in its immediate vicinity.

Such a condition prevails until from six to eight weeks after sowing—until, in fact, the bulb has begun to develop. At this stage dry-rot lesions become noticeable owing to death of occasional plants in the field, these showing rapid wilt and discoloration of the leaves. The crowns of such plants are completely rotted, although the bulbs may be quite healthy. Infection of these individual plants occurs from spores being washed from the leaves by way of the channels on the upper surfaces of the leaf-stalks to the crown. In the lesion thus produced numerous pycnidia are formed, and from these, during moist weather, spore tendrils are produced. These bulbs serve as the chief means whereby the disease carries over the earlier parts of the season, for the fungus can live in the bulb-tissues for several weeks, and, whenever the weather conditions are moist, during the whole of this period is capable of producing spores. These, as has been shown, are

embedded in mucilage. Now, as leaf-motion in the field is constant, leaves frequently brush these tendrils and in turn become infected; or the tendrils may thus be carried on to other surfaces and spores washed to the crowns of contiguous plants by rain. The significance of this primary bulb-infection is that infection centres, capable of infecting over a considerable period, are established over the field.

From these centres the disease spreads to contiguous plants in the rows, so that within a few weeks after scattered infected plants have

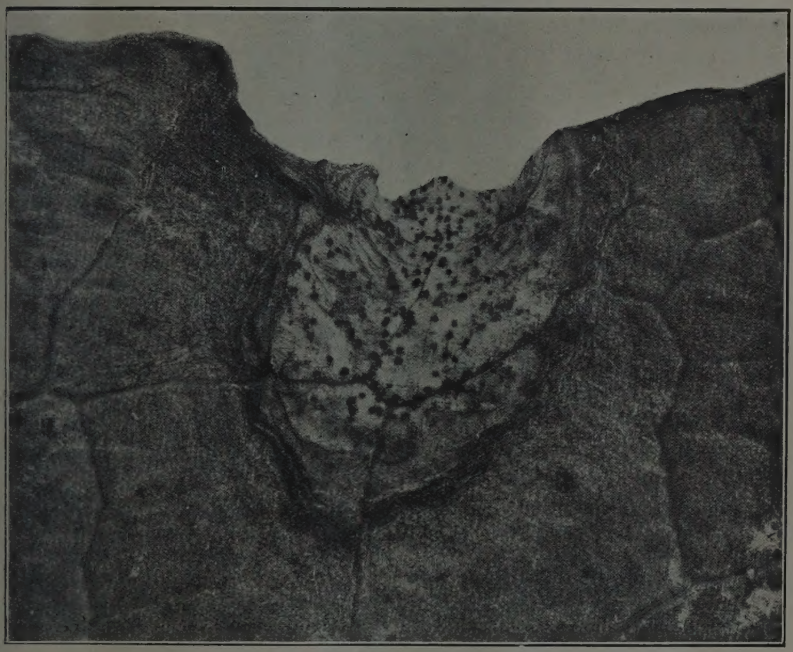

FIG. 4. LESION PRODUCED BY THE DRY-ROT ORGANISM IN SWEDE-LEAF. × 10.

The black bodies are the pycnidia.

[*Photo by H. Drake.*

become noticeable others in their vicinity show lesions, and in turn rot. It must be stressed that infection in the field is possible only where spores are brought into contact with the leaf or bulb. Such contact is effected by means of the leaves or by the spores (embedded in mucilage) being splashed to other plants in rain-drops. That is why infected plants at this or a later stage occur in the nature of small irregularly circular areas, and not simultaneously over the field. Wind plays only an indirect part in spore-dispersion—that is, by

moving the leaves about, or in blowing drops of rain containing spores to contiguous plants. It follows, therefore, that if one field is separated from another by a road or similar gap, the disease cannot be transmitted across such a gap, provided insects, animals, or cultural implements do not carry the disease from diseased to healthy plants across the intervening space. And as moisture is essential to spore-production, this explains why the disease is more severe in wet seasons or in wet or low-lying areas in the field; for under such conditions spore-production is continuous and infection can occur almost continuously. Conversely, dry-weather conditions inhibit infection; so that in certain localities of New Zealand, and in certain seasons throughout, little or no dry-rot is present.

From the small initial infection areas the disease gradually spreads outwards, so that frequently these infected areas merge and their individuality is lost. Should wet-weather conditions prevail, the disease may in the course of two or three months spread throughout the whole field; in fact, in the South it is not unusual to find in a season favourable to dry-rot 80 per cent., or even more, of the crop affected.

Initial bulb-infection is invariably followed by death of the plant. This is due to the fact that the diameter of the bulb is small and the lesion quickly destroys all tissues in its vicinity. Later, as the bulbs become considerably larger, the number of lesions present may be large, but each may penetrate to a slight extent only, so that the bulb, although with numerous lesions, may not decay for several months. Complete decay of large bulbs, as a rule, occurs only towards the end of the season, when they may dry out and assume a mummied appearance. Such mummied bulbs are a possible source of infection the following season, for unless they are buried they may lie on the ground (even when ploughed, as the cultural implements frequently bring them to the surface) until the next crop has appeared. This applies generally only when a crop has been fed off late (as in September) and a second crop of the same host sown early (as in November). The writer has shown in his bulletin that the disease may persist in these mummied bulbs for several months, but cannot persist in the soil in any other form.

Leaf-infection occurs of necessity first in the life-cycle of dry-rot. Later, when the bulbs are formed, bulb-infection is the more common; but at all stages leaf lesions may be found. Lesions develop chiefly on the neck and crown of the bulb, less commonly on the side (save where much exposed), and very seldom at or below ground-level. The explanation of this is that most of the spores are distributed by leaf-contact first with diseased plants, then to the healthy leaves or bulbs. The rarity of ground-level infection shows that seldom are the spores carried to this part, and this can be explained when the shape of the average bulb is considered. As has been shown, spores are washed down from the leaves to the crown and neck of the bulb, or deposited on the neck and crown directly by leaf-contact. If a bulb is normally upright the shoulder prevents the spores being washed to the lower portion; if, on the other hand, the bulb is inclined, the shoulder is no longer a protection, and heavy infection may result. Infections at or below ground-level are, of course, merely accidental.

FIG. 5. NECK-INFECTION OF SWEDE-BULB. NATURAL SIZE.
Note the complete destruction of all tissues in this region.
[*Photo by H. Drake.*

on account of their rarity; they occur merely through spores being deposited on the bulb at this point, those below ground-level occurring only when the soil has been forced away from the bulb, such as happens occasionally when the bulb has been moved by wind or by animals.

It has been shown that the disease is carried in the seed. It is now necessary to explain the manner in which the seed becomes

infected; and this has been facilitated by the liberal manner in which Mr. C. W. Body, of Kelso, Otago, has allowed us to make use of material from his swede-seed area.

FIG. 6. DRY-ROT INFECTION ON SWEDE SEED-PODS. NATURAL SIZE.

Seed removed from beneath these lesions produced typical dry-rot colonies on culture media.

[*Photo by H. Drake.*

The disease becomes established in bulbs to be used for seed in the usual manner. When the seed-spike develops, its leaves become infected, and the disease spreads from these to the spike. On both are produced abundant lesions, from which spores are produced. As the seed-pods develop, spores are carried to these, the fungus penetrates

FIG. 7. LESION PRODUCED IN SWEDE-BULB AS A RESULT OF EARLY INFECTION HALF NATURAL SIZE.

[Photo by E. B. Levy.

the pod and establishes an infection centre. From this the hyphæ of the fungus grow into the pod, and partially surround the seeds contained therein. The hyphæ grow directly into the seed-coat, and remain there in a quiescent condition until the seed has germinated, when, as has been shown, the fungus grows out from the seed-coat into the cotyledons, there producing lesions from which initial infections of the crop arise. The fungus may remain in a dormant condition in the seed for a considerable period—two or three seasons—but not indefinitely, for it has been established in this Laboratory that it tends to disappear from the seed after long keeping. For example, last season a line of 1926 seed harvested in 1925, containing 5 per cent. of diseased seed, was used extensively in experimental work. This season part of the same line was again used, and was found to contain only 0·1 per cent. diseased seed. This fact—that the disease cannot live indefinitely in the host—is already known with several other seed-carried diseases. It cannot be made use of in commercial control, however, as to keep a line sufficiently long for the disease to disappear would prove expensive, in that the germination percentage of the seed drops considerably each season. For example, the line with 5 per cent. of diseased seed last season gave a germination average of 85 per cent.; this season the germination had fallen to 54 per cent. It is not considered necessary to give here the process elaborated for detecting the presence of the disease in a line of seed; this has been dealt with fully in the bulletin.

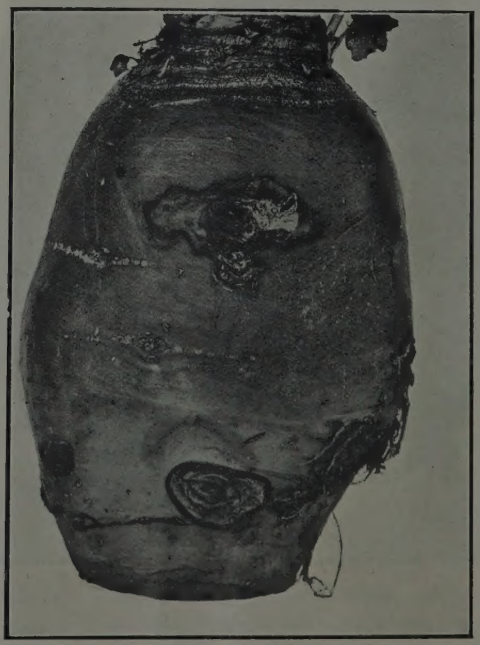

FIG. 8.

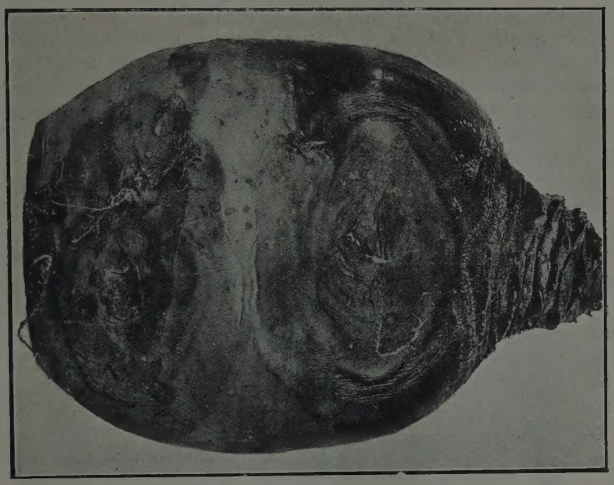

FIG. 9.

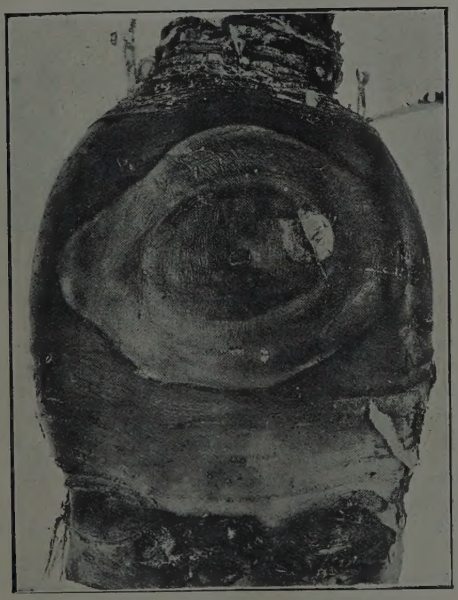

FIG. 10.

FIGS. 8, 9, 10. DIFFERENT TYPES OF LESIONS PRODUCED ON SWEDE-BULBS. HALF NATURAL SIZE.

[Photos by H. Drake.

Methods of Control of Dry-rot.

As dry-rot is seed-borne, it naturally follows that some method of treating the seed so as to destroy the fungus without affecting seed-germination would be the efficient method of combating it. Therefore a comprehensive series of experiments was conducted with the object of finding some such treatment. In these experiments it was found that the standard treatments for externally borne diseases were quite ineffective; and also that the standard methods for internal seed-borne diseases were equally ineffective. Finally, the following original method was found completely to eliminate the disease without material damage to the seed, *always provided this was initially of high-germination capacity*:—

Treatment: Half fill small cloth bags holding 2 lb. of seed or less; immerse these for one hour in a 0·25-per-cent. solution of Semesan held at 115° F. Ensure that seed is properly covered with the solution during this period. Remove bags, drain off surplus water, and spread seed in thin layers to dry (preferably on blotting-paper in a sunny place); or dry by passing through the seed (spread out in thin layers on cheese-cloth) a current of air heated to about 80° F.

In addition to using disease-free seed, or seed which has been treated so as to destroy the fungus, it is necessary that such be sown on land which has not been under swedes, turnips, or cabbages for twelve months. For, as has been shown, mummied bulbs may carry the disease for sufficiently long a period to cause infection of the following season's crop.

In numerous field experiments conducted during the past season it was found that the treated seed used did not suffer appreciably as a result of the treatment specified above, and in many of the crops the treatment appeared to have a decidedly stimulating effect. Further, the treatment proved quite satisfactory as a controllant of the disease where extraneous sources of infection were eliminated.

Unfortunately, it is not practicable under present conditions to treat the swede and turnip seed coming into New Zealand so as to free it from disease, for it has been found that, unless the initial germination of the seed is high, serious depreciation in germination follows treatment. Consequently, with the great bulk of seed coming into the Dominion, treatment would seriously affect germination, as the germination of the average line is not of the first order. Another factor operating against bulk treatment is the present method of handling seed. A number of lines are forwarded in sealed 1 lb. or 2 lb. bags. To open these, treat the seed, rebag, and reseal would entail an enormous amount of work, necessitating the employment of a considerable staff. And to treat the bulk seed would require the erection of a large plant and the employment of highly skilled specialists.

These factors, together with others of a technical nature—which need not be enumerated here—make it appear more reasonable to employ some other method of elimination of the disease from the seed. Two other and more practicable methods are open to consideration.

One of these is to eliminate the disease from the seed before it comes to this country; the other, to grow in New Zealand disease-free seed.

To eliminate the disease from the seed before it comes to New Zealand seems to be, for the time at least, the more reasonable method, as then use could be made of the available machinery for distribution, and competition with the British seedsmen avoided. If swede- and turnip-seed growing were practised in the Dominion (and there is nothing to prevent its being a success under proper management) competition with seedsmen and merchants would be inevitable—a condition which could be met only by legislation.

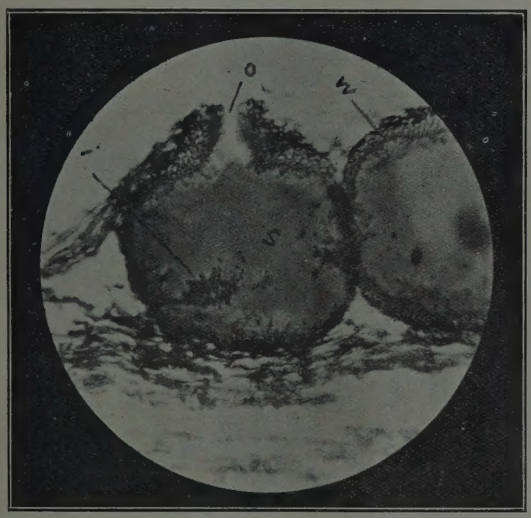

FIG. 11. SECTION OF PYCNIDIUM OF THE CAUSAL ORGANISM. × 125.

[*Photo by Writer.*

In Britain (which supplies all but a few hundredweight of our annual requirements) most of the bulk seed is grown by farmers under contract to the seedsmen. The latter supply pedigree seed, raised for the most part on their own farms from selected bulbs, to the growers, who in turn sow it either directly in the fields, or else first sow in seed-beds or boxes and transplant the seedlings when they have reached a stage when the bulb is about the thickness of a lead-pencil. Sowing or transplanting is carried out in early autumn (August), and the crop harvested about twelve months later. The seed-spikes are cut and handled just as are oats or wheat, and, when dry, are stacked in long ricks until ready for threshing. A good average yield of turnip-seed is about 1,800 lb. per acre. Now, as we imported 564,480 lb. of swede and turnip seed in 1926, a rough calculation will show that to produce this amount, with an average

yield of 1,500 lb. per acre, 375 lb. of seed (here termed "mother seed") would require to be sown. To treat this amount would prove a relatively simple matter; and, what is of greater moment, even were germination reduced by 50 per cent., the loss incurred would be slight compared with the same loss were the bulk seed treated. One other point of interest is worthy of mention here. The seed coming to New Zealand is at least one year old before it reaches the farmer; consequently, unless of the finest quality, its germination will have been reduced by keeping by from 5 to 15 per cent. Such seed, if treated, may suffer considerable reduction in germination here because of this initial reduction, but would suffer little damage if treated twelve months previously.

Thus, if the seedsmen in Britain were to treat their "mother seed" before it was issued to the contract growers, and if they were in addition to ensure that such was grown on land which had not been under brassicas for twelve months previously, a clean crop would result; for, as has been shown, infection can occur only from contiguous diseased plants, the spores of the fungus not being wind-borne. A clean crop would result in the seed being free from the disease, would result in increased seed-yields, and would eliminate the necessity for our taking any further steps in New Zealand (apart from ordinary farm-management) to deal with dry-rot.

Were such practices adopted in Britain, in a season or two dry-rot might be entirely eliminated from the swede and turnip crops of New Zealand. It appears (to the writer at least) reasonable that this method of fighting dry-rot should be given a fair trial, as then the British seedsmen, who have worked so long to produce the fine strains of swedes and turnips of to-day, would, if they adopted the methods outlined, suffer no appreciable loss, but would benefit in material increase in yields, and particularly in retaining the New Zealand trade. If, on the other hand, growing the seed in New Zealand were resorted to, and clean seed produced, it would be necessary to protect such a developing industry, and prevent the introduction into the Dominion of diseased seed by legislating against the importation of any lines carrying the disease. It would not be possible to enforce such legislation until the machinery for growing and distributing such seed were in operation—a matter requiring several years at least—otherwise we should be faced with a seed famine, for, judging by the samples of 1926 seed tested, few lines would gain admittance were such measures enforced.

Finally, it should be noted by the farmer that on being supplied with clean seed it would be necessary for him to undertake certain simple precautions. These are—

(1) To abstain from sowing swedes or turnips on land which was in a similar crop the previous season.

(2) To clear absolutely all previous season's seed from his drills, &c.

(3) To destroy *any* previous season's swede or turnip seed on his property.

(4) To refuse (when disease-free seed is procurable) to buy from any merchant seed which has not been certified as being disease-free by the Department of Agriculture.